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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
10/574,422	11/07/2006	Eggert Stockfleth	50125/084002	7550

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EXAMINER	
MI, QIUWEN	
ART UNIT	PAPER NUMBER
1655	

NOTIFICATION DATE	DELIVERY MODE
12/08/2008	ELECTRONIC

Please find below and/or attached an Office communication concerning this application or proceeding.

The time period for reply, if any, is set in the attached communication.

Notice of the Office communication was sent electronically on above-indicated "Notification Date" to the following e-mail address(es):

patentadministrator@clarkelbing.com

Office Action Summary	Application No.	Applicant(s)	
	10/574,422	STOCKFLETH, EGGERT	
	Examiner	Art Unit	
	QIUWEN MI	1655	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE *3* MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *21 October 2008*.

2a)☒ This action is **FINAL**. 2b)☐ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *1,3-6 and 8-36* is/are pending in the application.

4a) Of the above claim(s) *33 and 34* is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *1, 3-6, 8-32, 35, and 36* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☒ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☐ Information Disclosure Statement(s) (PTO/SB/08) Paper No(s)/Mail Date _____.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____ .
5) ☐ Notice of Informal Patent Application
6) ☐ Other: _____.

DETAILED ACTION

Applicant's amendment in the reply filed on 10/21/08 is acknowledged, with the cancellation of Claims 2 and 7; and the additional newly added Claim 36. Claims 1, 3-6, and 8-36 are pending. Claims 33 and 34 are withdrawn from consideration. **Claims 1, 3-6, 8-32, 35, and 36 are examined on the merits.**

Any rejection that is not reiterated is hereby withdrawn.

Claim Rejections –35 USC § 102

The following is a quotation of the appropriate paragraphs of 35 U.S.C. 102 that form the basis for the rejections under this section made in this Office action:

A person shall be entitled to a patent unless –

(b) the invention was patented or described in a printed publication in this or a foreign country or in public use or on sale in this country, more than one year prior to the date of application for patent in the United States.

Claims 1, 3, 4, 6, 8-20, and 29 remain rejected under 35 USC § 102 (b) as being anticipated by Bickers et al (Novel approaches to chemoprevention of skin cancer, The Journal of Dermatology, 27: 691-695, 2000), as evidenced by Dou et al (US 2002/0151582)*.

This rejection is maintained for reasons of record set forth in the Office Action mailed out on 6/17/2008, repeated below. Applicants' arguments filed have been fully considered but they are not deemed to be persuasive.

Bickers et al teach that "we demonstrated that green tea, black tea and constituent polyphenols protect against chemical- and ultraviolet B (UVB)-induced carcinogenesis and reduce the growth of established tumors in skin" (see Abstract). Bickers et al also teach that "we have found that oral administration of a standardized green tea extract (SGTE) prior to and during treatment of SKH-1 mice diminished PUVA-induced skin hyperplasia and hyperkeratosis (pre-cancerous sin lesion)" (see Abstract); "Topical application of SGTE to human skin prior to PUVA-treatment inhibited the delayed skin inflammatory response"; "Similarly, oral and topical administration of standardized black tea extract and its two major polyphenolic sub-fractions protect against UVB-induced erythema in SKH-1 mice" (see Abstract). Bickers et al state that "both black and green tea and their constituents inhibit the tumor initiation, promotion and malignant progression stages of multi-step skin carcinogenesis" (page 693, 1st column, 2nd paragraph). Bickers et al indicate that "several experimental studies conducted in human skin have shown the efficacy of tea constituents as inhibitors of carcinogenesis-associated surrogate markers or inflammation (page 693, 2nd column, 2nd paragraph). Bickers et al conclude that "we also observed that topically applied green tea is effective in abrogating PUVA-induced inflammatory responses in human skin" (page 693, 2nd column, 2nd paragraph).

As evidenced by Dou et al (US 2002/0151582), green tea contains polyphenol compounds EGCG (formula I and II in claims 13 and 14 are thus met), ECG, GCG, or CG (claim 3).

Therefore, the reference is deemed to anticipate the instant claim above.

Applicant argues that "Bickers does not anticipate the present claims because this reference fails to teach a method for treating a precancerous lesion of the skin by administering a pharmaceutically effective amount of a polyphenol to a patient, as required by claim 1. In contrast to the claimed methods, Bickers instead teaches the prevention of a precancerous lesion, a result far different from treatment of an already established precancerous condition. This distinction is first highlighted by the title of Bickers, "Novel Approaches to Chemoprevention of Skin Cancer" (emphasis added). Moreover, throughout Bickers, prevention or protection against precancerous lesions is repeatedly discussed.

Applicant argues that "Turning specifically to the passages cited by the Office, Applicant notes that each of these teachings similarly refers to either the protective effect of standardized green tea extract or the treatment of cancerous tumors. The Examiner first focuses on a passage (Office action, page 3 and Abstract of Bickers) that states "green tea, black tea and constituent polyphenols protect against chemical- and ultraviolet B (UVB)-induced carcinogenesis and reduce the growth of established tumors in skin" (emphasis added). This is consistent with Applicant's characterization of the reference as failing to treat precancerous skin lesions" (page 12, 3rd paragraph).

Applicant argues that "The Examiner further notes that "standardized green tea extract (SGTE) prior to and during treatment of SKH-1 mice diminished PUVA-induced skin hyperplasia and hyperkeratosis (precancerous sin *(sic)* lesion)" (Office action, page 3 and Abstract of Bickers). This statement is based on the result in Bickers that "oral administration of

green tea extract prior to or during multiple PUVA treatments of SKH-1 hairless mice reduces hyperplasia, hyperkeratosis, erythema and edema" (page 693, right column of Bickers, under "Anti-Inflammatory Activity of Tea Extracts"). Again, the green tea extract is preventing PUVA damage, and not treating an established lesion. As noted specifically by Bickers when describing these results, the administration of a green tea extract prior to PUVA treatment or during PUVA treatment provides only a protective effect against such conditions (page 693, left column of Bickers, under "Anti- Inflammatory Activity of Tea Extracts"). Indeed, in none of the above passages does Bickers indicate that the extracts provide a therapeutic effect" (page 12, last paragraph).

Applicant argues that "The Examiner also notes (Office action, page 3) that Bickers teaches that "[t]opical application of SGTE to human skin prior to PUVA-treatment inhibited the delayed skin inflammatory response" (emphasis added). This statement, however, again refers to a protective effect. The same is true of the citation (Office action, page 3) that "oral and topical administration of standardized black tea extract and its two major polyphenolic sub-fractions protect against UVB-induced erythema in SKH-1 mice" (emphasis added)" (page 13, 1st paragraph).

Applicant argues that "Furthermore, the Office cites experimental studies that have shown the efficacy of tea constituents as inhibitors of carcinogenesis-associated surrogate markers or inflammation (page 4 and page 693, right column of Bickers, under Anticarcinogenic Effects of Tea Extracts in Humans"). As reported by Bickers, pretreatment of the back skin of normal human volunteers protected against UVB (2 MED)-mediated induction of sunburn-cell formation and depletion of CD 1 a+ Langerhans cells. Bickers concludes that

"topically applied green tea is effective in abrogating PUVA-induced inflammatory responses in human skin" (page 693, right column of Bickers, under "Anticarcinogenic Effects of Tea Extracts in Humans"). This statement again fails to address treatment of established precancerous lesions, relating instead to a protective effect of the green tea extract against UV damage" (page 13, 2nd paragraph).

This is not found persuasive. It is true that Bickers teaches the chemoprevention of skin cancer in many places in the reference. However, Bickers also explicitly teaches "oral administration of green tea extract prior to or during multiple PUVA treatments of SKH-1 hairless mice reduces hyperplasia, hyperkeratosis, erythema and edema". Although, skin cancer is not established during PUVA treatment, hyperplasia, hyperkeratosis, erythema (redness of skin), or edema occur during PUVA treatment process. In addition, reduce the incidence of developing skin cancer from precancerous lesion hyperplasia, hyperkeratosis, erythema (redness of skin), and edema itself is a treatment of precancerous lesion. In the instance case, skin cancer is prevented since the precancerous lesions hyperplasia, hyperkeratosis, erythema (redness of skin), and edema got treated by green tea extract.

Applicant argues that "Finally, Applicant points to a statement made in Bickers and cited by the Examiner, that "both black and green tea and their constituents inhibit the tumor initiation, promotion and malignant progression stages of multi-step skin carcinogenesis" (page 693, left column of Bickers, under "Anticarcinogenic Effects of Tea Extracts in Animal Models"). This passage of Bickers cites Mukhtar et al. *(Toxicological Sciences*

52" 111-117, 1999), provided herewith, as describing the source of this result. But, as in Bickers, Mukhtar describes only chemoprevention of cancer by green tea, and not the treatment of a precancerous lesion. Specifically, Mukhtar states: [W]e showed that EGCG induces apoptosis and cell cycle arrest in human epidermoid carcinoma (A431) cells (Ahmad *et al.,*1997). Importantly, this apoptotic response was specific for cancer cells, since EGCG treatment also resulted in the induction of apoptosis in human carcinoma keratinocytes HaCaT, human prostate carcinoma cells DU145, and mouse lymphoma cells LY-
R, but not for normal human epidermal keratinocytes. (Page 113, right column of Mukhtar; emphasis added) (page 13, last two paragraphs bridging page 14).

Applicant argues that "Thus, as described in Mukhtar (citing Ahmad et al. *(Journal of the National Cancer Institute* 89:1881-1886, 1997, provided herewith), treatment with epigallocatechin-3-gallate stimulated apoptosis of HaCaT, L5178Y, and DU 145 cells, but not of normal human epidermal keratinocytes (see, e.g., the abstract of Ahmad). Thus, Mukhtar and Ahmad, the references cited by Bickers, clarify that the statement relied upon by the Office relates only to cancerous cells, and teaches nothing about the effects of the administration of a polyphenol on precancerous cells (e.g., the skin cells of claim 1). The other reference cited by the Examiner, Dou, does not relate to treatment of precancerous lesions. Therefore, Bickers, alone or as evidenced by Dou, fails to teach all the elements of claims 1, 3, 4, 6, 8-20, and 29, and Applicant respectfully requests that this rejection of the claims under 35 U.S.C. § 102 be withdrawn" (page 14, 2nd paragraph).

This is not found persuasive. Since precancerous lesion is not normal human epidermal keratinocytes, thus the attached Mukhtar reference is not meaningful in rebutting the rejection as

the fact that epigallocatechin-3-gallate does not stimulate apoptosis of normal human epidermal keratinocytes has nothing to do with whether or not epigallocatechin-3-gallate can treat precancerous lesion.

Claim Rejections –35 USC § 103

The following is a quotation of 35 U.S.C. 103(a) which forms the basis for all obviousness rejections set forth in this Office action:

(a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negatived by the manner in which the invention was made.

Claims 1, 3-6, 8-26, and 29 remain rejected under 35 U.S.C. 103(a) as being unpatentable over Bickers et al (Novel approaches to chemoprevention of skin cancer, The Journal of Dermatology, 27: 691-695, 2000), as evidenced by Dou et al (US 2002/0151582)*.

This rejection is maintained for reasons of record set forth in the Office Action mailed out on 6/17/2008, repeated below. Applicants' arguments filed have been fully considered but they are not deemed to be persuasive.

Bickers et al teach that "we demonstrated that green tea, black tea and constituent polyphenols protect against chemical- and ultraviolet B (UVB)-induced carcinogenesis and reduce the growth of established tumors in skin" (see Abstract). Bickers et al also teach that "we have found that oral administration of a standardized green tea extract (SGTE) prior to and during treatment of SKH-1 mice diminished PUVA-induced skin hyperplasia and hyperkeratosis

(pre-cancerous sin lesion)" (see Abstract); "Topical application of SGTE to human skin prior to PUVA-treatment inhibited the delayed skin inflammatory response"; "Similarly, oral and topical administration of standardized black tea extract and its two major polyphenolic sub-fractions protect against UVB-induced erythema in SKH-1 mice" (see Abstract). Bickers et al state that "both black and green tea and their constituents inhibit the tumor initiation, promotion and malignant progression stages of multi-step skin carcinogenesis" (page 693, 1st column, 2nd paragraph). Bickers et al indicate that "several experimental studies conducted in human skin have shown the efficacy of tea constituents as inhibitors of carcinogenesis-associated surrogate markers or inflammation (page 693, 2nd column, 2nd paragraph). Bickers et al conclude that "we also observed that topically applied green tea is effective in abrogating PUVA-induced inflammatory responses in human skin" (page 693, 2nd column, 2nd paragraph).

As evidenced by Dou et al (US 2002/0151582), green tea contains polyphenol compounds EGCG (formula I and II in claims 13 and 14 are thus met), ECG, GCG, or CG (claim 3).

Bickers et al do not teach the amount of the polyphenols in the composition.

Therefore, it would have been *prima facie* obvious for one of ordinary skill in the art at the time the invention was made to use the inventions of Bickers et al since they provide scientific data for novel approaches to chemoprevention of skin cancer, one of ordinary skill in the art would have been motivated to make the modifications. The result-effective adjustment in conventional working parameters (e.g., determining an appropriate amount of the each polyphenol components as claimed isolated from green tea within the composition) is deemed merely a matter of judicious selection and routine optimization which is well within the purview of the skilled artisan.

Since Bickers et al teach administering green tea extract (SGTE) prior to and during treatment of SKH-1 mice diminished PUVA-induced skin hyperplasia and hyperkeratosis, and hyperkeratosis is not a hyperplasia, Condyloma acuminate, warts, and cervical intra-epithelial neoplasia, thus the limitation of claim 5 is met.

From the teachings of the references, it is apparent that one of the ordinary skills in the art would have had a reasonable expectation of success in producing the claimed invention.

Thus, the invention as a whole is *prima facie* obvious over the references, especially in the absence of evidence to the contrary.

Claims 1, 3-6, 8-32, and 35 remain rejected under 35 U.S.C. 103(a) as being unpatentable over Bickers et al (Novel approaches to chemoprevention of skin cancer, The Journal of Dermatology, 27: 691-695, 2000), in view of Brash et al (US 2002/0198161), and further in view of Voet (US 6,723,750), as evidenced by Dou et al (US 2002/0151582)*.

This rejection is maintained for reasons of record set forth in the Office Action mailed out on 6/17/2008, repeated below. Applicants' arguments filed have been fully considered but they are not deemed to be persuasive.

Bickers et al teach that "we demonstrated that green tea, black tea and constituent polyphenols protect against chemical- and ultraviolet B (UVB)-induced carcinogenesis and reduce the growth of established tumors in skin" (see Abstract). Bickers et al also teach that "we have found that oral administration of a standardized green tea extract (SGTE) prior to and during treatment of SKH-1 mice diminished PUVA-induced skin hyperplasia and hyperkeratosis

(pre-cancerous sin lesion)" (see Abstract); "Topical application of SGTE to human skin prior to PUVA-treatment inhibited the delayed skin inflammatory response"; "Similarly, oral and topical administration of standardized black tea extract and its two major polyphenolic sub-fractions protect against UVB-induced erythema in SKH-1 mice" (see Abstract). Bickers et al state that "both black and green tea and their constituents inhibit the tumor initiation, promotion and malignant progression stages of multi-step skin carcinogenesis" (page 693, 1st column, 2nd paragraph). Bickers et al indicate that "several experimental studies conducted in human skin have shown the efficacy of tea constituents as inhibitors of carcinogenesis-associated surrogate markers or inflammation (page 693, 2nd column, 2nd paragraph). Bickers et al conclude that "we also observed that topically applied green tea is effective in abrogating PUVA-induced inflammatory responses in human skin" (page 693, 2nd column, 2nd paragraph).

As evidenced by Dou et al (US 2002/0151582), green tea contains polyphenol compounds EGCG (formula I and II in claims 13 and 14 are thus met), ECG, GCG, or CG (claim 3).

Bickers et al do not teach additive isopropyl myristate, form of ointment, or combined with different treatment curettage, or the claimed amount of the polyphenols.

Brash et al teaches that skin precancers are being treated, the preferred mode of administration is topical. The topical application may contain carrier, excipient or vehicle ingredients such as isopropyl myristate etc., and mixtures thereof to form lotions, creams, emulsions, gels, or ointments [0086].

Voet teaches that the current management options for visible or easily perceived and diagnosed precancerous dermatological lesions such as Aks (thus claim 35 is met) include cryosurgery with liquid nitrogen, topical treatment, and curettage (col 2, lines 15-20). Voet also teaches that curettage, which involves the use of a curette to scrape away the lesion, is another common method of treatment for easily perceptible precancerous skin lesions. The primary advantage of curettage is the ability to submit the specimen for histologic analysis.

Therefore, it would have been *prima facie* obvious for one of ordinary skill in the art at the time the invention was made to use the carrier isopropyl myristate and ointment form of Brash et al, and the treatment of curettage from Voet in the current invention since carrier isopropyl myristate and ointment form are the conventional carrier and pharmaceutical form that have been used successfully in treating precancerous lesions in the topical route according to Brash et al; and combining the treatment curettage from Voet with the topical could monitor the histologic status of the tissue treated by topical administration. Since both Brash et al, and the treatment of curettage from Voet yielded beneficial results in treating precancerous lesions, one of ordinary skill in the art would have been motivated to make the modifications. The result-effective adjustment in conventional working parameters (e.g., determining an appropriate amount of the each polyphenol components as claimed isolated from green tea within the composition) is deemed merely a matter of judicious selection and routine optimization which is well within the purview of the skilled artisan.

Since Bickers et al teach administering green tea extract (SGTE) prior to and during treatment of SKH-1 mice diminished PUVA-induced skin hyperplasia and hyperkeratosis, and

hyperkeratosis is not a hyperplasia, Condyloma acuminate, warts, and cervical intra-epithelial neoplasia, thus the limitation of claim 5 is met.

From the teachings of the references, it is apparent that one of the ordinary skills in the art would have had a reasonable expectation of success in producing the claimed invention.

Thus, the invention as a whole is *prima facie* obvious over the references, especially in the absence of evidence to the contrary.

*This reference is cited merely to relay an intrinsic property and is not used in the basis for rejection *per se.*

Claim 36 is newly rejected under 35 U.S.C. 103(a) as being unpatentable over Bickers et al (Novel approaches to chemoprevention of skin cancer, The Journal of Dermatology, 27: 691-695, 2000), in view of An Kathy et al (Cyclooxygenase-2 expression in murine and human nonmelanoma skin cancers: implications for therapeutic approaches, Photochemistry and photobiology, (2002 Jul) Vol. 76, No. 1, pp. 73-80), as evidenced by Dou et al (US 2002/0151582)*.

This is a new rejection necessitated by the Applicant's amendment filed on 10/21/08.

Bickers et al teach that "we demonstrated that green tea, black tea and constituent polyphenols protect against chemical- and ultraviolet B (UVB)-induced carcinogenesis and reduce the growth of established tumors in skin" (see Abstract). Bickers et al also teach that "we have found that oral administration of a standardized green tea extract (SGTE) prior to and during treatment of SKH-1 mice diminished PUVA-induced skin hyperplasia and hyperkeratosis (pre-cancerous sin lesion)" (see Abstract); "Topical application of SGTE to human skin prior to

PUVA-treatment inhibited the delayed skin inflammatory response"; "Similarly, oral and topical administration of standardized black tea extract and its two major polyphenolic sub-fractions protect against UVB-induced erythema in SKH-1 mice" (see Abstract). Bickers et al state that "both black and green tea and their constituents inhibit the tumor initiation, promotion and malignant progression stages of multi-step skin carcinogenesis" (page 693, 1st column, 2nd paragraph). Bickers et al indicate that "several experimental studies conducted in human skin have shown the efficacy of tea constituents as inhibitors of carcinogenesis-associated surrogate markers or inflammation (page 693, 2nd column, 2nd paragraph). Bickers et al conclude that "we also observed that topically applied green tea is effective in abrogating PUVA-induced inflammatory responses in human skin" (page 693, 2nd column, 2nd paragraph).

As evidenced by Dou et al (US 2002/0151582), green tea contains polyphenol compounds EGCG (formula I and II in claims 13 and 14 are thus met), ECG, GCG, or CG (claim 3).

Bickers et al do not teach treating actinic keratosis.

An Kathy et al teach COX-2 expression was also increased in human actinic keratoses. An Kathy et al also teach acute exposure of the human skin to UVB (minimum erythema dose x 4) caused a transient enhancement of the COX-2 expression, which reverted to baseline within hours; however, in murine skin the expression persisted for several days. Pretreatment with the topically applied green tea extract (1 mg/cm2) largely abrogated the acute COX-2 response to UVB in mice or humans. In summary, enhanced COX-2 expression serves as a marker of epidermal UVB exposure for murine and human NMSC. These results suggest that COX-2 inhibitors could have potent anticarcinogenic effects in UVB-induced skin cancer (see Abstract).

It would have been *prima facie* obvious for one of ordinary skill in the art at the time the invention was made to use the green tea extract to treat actinic keratoses from An Kathy et al since An Kathy et al teach green tea extract (1 mg/cm2) largely abrogated the acute COX-2 response to UVB in mice or humans. Since both Bickers et al and An Kathy et al teach using green tea extract to treat precancerous lession, one of ordinary skill in the art would have been motivated to make the modifications and combine the two references together.

Since Bickers et al teach administering green tea extract (SGTE) prior to and during treatment of SKH-1 mice diminished PUVA-induced skin hyperplasia and hyperkeratosis, and hyperkeratosis is not a hyperplasia, Condyloma acuminate, warts, and cervical intra-epithelial neoplasia, thus the limitation of claim 5 is met.

From the teachings of the references, it is apparent that one of the ordinary skills in the art would have had a reasonable expectation of success in producing the claimed invention.

Thus, the invention as a whole is *prima facie* obvious over the references, especially in the absence of evidence to the contrary.

*This reference is cited merely to relay an intrinsic property and is not used in the basis for rejection *per se.*

Conclusion

No claim is allowed.

Applicant's amendment necessitated the new ground(s) of rejection presented in this Office action. Accordingly, **THIS ACTION IS MADE FINAL**. See MPEP § 706.07(a). Applicant is reminded of the extension of time policy as set forth in 37 CFR 1.136(a).

A shortened statutory period for reply to this final action is set to expire THREE MONTHS from the mailing date of this action. In the event a first reply is filed within TWO MONTHS of the mailing date of this final action and the advisory action is not mailed until after the end of the THREE-MONTH shortened statutory period, then the shortened statutory period will expire on the date the advisory action is mailed, and any extension fee pursuant to 37 CFR 1.136(a) will be calculated from the mailing date of the advisory action. In no event, however, will the statutory period for reply expire later than SIX MONTHS from the date of this final action.

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Qiuwen Mi whose telephone number is 571-272-5984. The examiner can normally be reached on 8 to 5.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Terry McKelvey can be reached on 571-272-0775. The fax phone number for the organization where this application or proceeding is assigned is 571-273-8300.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see http://pair-direct.uspto.gov. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free). If you would like assistance from a USPTO Customer Service Representative or access to the automated information system, call 800-786-9199 (IN USA OR CANADA) or 571-272-1000.

QM

/Michael V. Meller/

Primary Examiner, Art Unit 1655